

FLUID FLOW IN THE OSTEOCYTE ENVIRONMENT: A PARAMETRIC STUDY

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Introduction

There is increasing evidence of variation in osteocyte and lacuna morphology being associated with mechanical stimulation and disease state. Elongated osteocytes and lacunae have been found in lamellar long bone with a predominate direction of load [1,2]. More spherical lacunae have been found in flat [2], woven [3], immature [4] bone that does not have a predominate loading direction, but also in osteopenic [1,5] and osteoarthritic [5-8] long bone. The objective of this research is to use computational methods to predict the effect of osteocyte and lacuna morphology on the strain environment of the osteocyte to understand the functional significance of changes in their shape.

Methods

Three solid models of idealised bone-osteocyte interaction were developed in SolidWorks (Dassault Systemes, Tennessee). For each osteocyte lacuna type the ratio between minor and major axes, λ , was assumed to be 0.6, 0.25 and 1 (spherical), respectively. The geometry of the osteocyte was offset from each lacuna geometry allowing for an interstitial fluid (IF) space of 0.75 μm in thickness and the canaliculi were represented as channels with a diameter of 0.6 μm . The extra cellular matrix (ECM) was modelled as a 20 μm side cube [1]. Booleans operations were used to obtain the IF domain. ECM, Osteocyte and IF were then imported into Abaqus (V. 6.14 Simulia, Providence, RI) and meshed with tetrahedral elements for a total of 3 M elements for each cell type. All solid structures were modelled as linear elastic and isotropic (Table 1). The properties of IF were assumed to be similar to salt water [7]. A sinusoidal displacement boundary of 500 μm (frequency, $f = 1\text{Hz}$) was applied uniformly on the top surface of the ECM. To prevent rigid body motion, the opposing face was constrained symmetrically. An inlet pressure of 300 Pa ($f = 1\text{Hz}$) was assigned to the inlet on one face and the remaining inlets were defined as outlets at a relative pressure of 0 Pa. Fluid Structure Interaction (FSI) simulations were conducted allowing for a strongly coupled solution between solid and fluid domains.

	Young Modulus, E	Poisson Ratio, ν
ECM	16 GPa	0.38
Osteocyte	4.47 KPa	0.3

Table 1: Material properties of the extracellular matrix (ECM) and Osteocyte [6].

Results

Our results obtained from the spherical osteocyte model show that regions of highest velocities are located within osteocyte canaliculi, with maximum velocities of 200 $\mu\text{m/s}$ when the maximum displacement and pressure are applied to the model ($t = 0.25\text{ s}$). In contrast, fluid velocities are much lower surrounding the osteocyte cell body, with magnitudes of approximately 50 $\mu\text{m/s}$. Consistently, the strain on the surface of the spherical osteocyte resulting from the interstitial fluid flow is higher along the canaliculi with maximum value of 1000 $\mu\epsilon$. Most of the cell membrane, on the other hand, experiences strain value of at least one order less.

Discussion

The purpose of this parametric study is to predict the effect of osteocyte and lacuna morphology on their mechanical environment with the ultimate aim to understand the mechanical pathways responsible for the osteocyte shape adaptation. In this regard, we have implemented FSI simulations and investigated the complex interaction between solid and fluid phases for a spherical cell geometry, which results are in agreement with our previous study and the literature [1, 6-8]. We are currently running the models for the more elongated geometries. However, the idealized models considered for this work can only provide indications of the relative strain map sensed by cells with different shape but not be informative on the absolute strain values.

References

1. Carriero A. et al, Bone, 2014, 61:116-24.
2. Vatsa A. et al, Bone, 43:452-8, 2008.
3. Hernandez C.J. et al, Bone, 35:1095-9, 2004.
4. Sugawara Y. et al, Bone, 52:189-96, 2013.
5. Van Hove R.P. et al, Bone, 45:321-9, 2009.
6. Verbruggen S.W. et al, J R Soc Interface, 9:2735-44, 2012.
7. Verbruggen S.W. et al, Biomech Model Mechanobiol, 13:85-97, 2014.
8. Verbruggen S.W. et al, Biophysical Journal, 108:1587-1598, 2015.

Acknowledgements

National Science Foundation 1829310
NIH grants 1R01HL136431, 1SC1DK103362, NSF grants CMMI-1662970, CMMI-1333560, MRI-0723027, MRI-1229449 and NYS DOH grant C31291GG

